

8.8.14 Trichomoniasis 1475

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8.8.14 Trichomoniasis 1475 Africa with pentavalent antimonials for 30–60 days or liposomal amphotericin B for 20 days. Supportive treatment Intercurrent infection must be sought and treated, and nutritional deficiencies corrected. Blood transfusion is rarely needed. Response to treatment Fever, splenic size, haemoglobin, serum albumin, and body weight are useful monitors of progress. Proof of parasitological cure is not usually necessary. Reassessment at 6 weeks and 6 months will detect over 90% of relapses. Serology is unhelpful in monitoring progress. Relapse rates should be under 4%. Relapsed patients are slower to respond and run a 40% chance of further relapses and of becoming unresponsive to antimony. Economic impact Visceral leishmaniasis is a major economic burden on affected families. The direct costs of an episode of visceral leishmaniasis in rural India or Bangladesh, where the drug is, in principle, provided free, are equivalent to the household's annual income. Prevention and control of cutaneous and visceral leishmaniasis Prevention is a matter of controlling reservoir hosts and sandfly vectors or of avoiding bites by vectors. Successful control requires an accurate knowledge of transmission in each ecological focus. In the Old World, urban cutaneous leishmaniasis is controlled by case-finding and treatment, better housing, and domestic spraying with residual insecticides, while rural leishmaniasis is controlled in the Middle East and North Africa by poisoning or destruction of gerbil colonies. Mediterranean and urban visceral leishmaniasis in South America can be controlled by the destruction or treatment of dogs, but dogs are infectious to flies before they become symptomatic and screening of dogs is problematic. Dog collars impregnated with permethrin reduce the numbers of flies that become infected. In India, mass campaigns to spray houses and cattle sheds are needed in addition to case-finding and treatment. In the interepidemic period, cases of PKDL should be sought and treated. Currently no nation has an effective control programme in place. In endemic populations, infection can be prevented during the season of transmission by the use of insect repellent creams and of fine mesh bed nets, top sheets or chadors (women's outer garments or cloaks) impregnated with permethrin during the hours of biting, usually around dusk and dawn. In endemic foci, a higher level of education in households is associated with lower rates of disease. Vaccines have proved disappointing. FURTHER READING Blum J, et al. (2014). LeishMan recommendations for treatment of cutaneous and mucosal leishmaniasis in travellers. *J Trav Med*, 21, 116–29. den Boer M, Davidson RN (2006). Treatment options for visceral leishmaniasis. *Expert Rev Anti Infect Ther*, 4, 187–97. Desjeux P (2001). The increase in risk factors for leishmaniasis world-wide. *Trans R Soc Trop Med Hyg*, 95, 239–43. Lockwood DNJ, Sundar S (2006). Serological tests for visceral leishmaniasis. *Br Med J*, 333, 711–12. Murray HW, et al. (2005). Advances in leishmaniasis. *Lancet*, 366, 1561–77. Sundar S, et al. (2011) Comparison of short-course multidrug treatment with standard therapy for visceral leishmaniasis in India:

an open-label, non-inferiority, randomised controlled trial. *Lancet*, 377, 477–86. World Health Organization (WHO) (2010). Control of the leishmaniasis: report of a meeting of the WHO Expert Committee on the Control of Leishmaniasis. Geneva, 22–26 March 2010. WHO technical report series; no. 949. World Health Organization, Geneva. Websites Centres for Disease Control. <http://www.cdc.gov/parasites/leishmaniasis/index.html> World Health Organization (WHO). Leishmaniasis. <http://www.who.int/leishmaniasis/en/> 8.8.14 Trichomoniasis Jane Schwebke

ESSENTIALS *Trichomonas vaginalis* is a sexually transmitted protozoan pathogen that may cause more than one-half of all curable sexually transmitted genital infections worldwide. Women with trichomoniasis are often asymptomatic, but they may develop vaginal malodour, discharge, erythema, or itching, and their male or female sexual partners may also be infected, although urethritis in men is less likely to cause symptoms. Women with trichomoniasis have an increased risk of HIV acquisition, HIV shedding, pelvic inflammatory disease, and preterm birth. For diagnosis, rapid antigen detection, culture, and polymerase chain reaction methods have advantages over conventional microscopy, but are more expensive. Oral metronidazole is usually an effective treatment, with both sexual partners needing to be treated to prevent reinfection. Introduction Trichomoniasis is an infection of the human urogenital tract caused by the flagellated protozoan *Trichomonas vaginalis*. There are about 170 million new cases each year, making it the world's most common Acknowledgement: The editors acknowledge the inclusion of material from Dr Sharon Hillier's chapter in the previous edition of the Oxford Textbook of Medicine.

section 8 Infectious diseases 1476 nonviral sexually transmitted infection and, according to the World Health Organization (WHO), it accounts for more than one-half of all curable sexually transmitted infections worldwide. Pregnant women who have trichomoniasis are at increased risk of preterm delivery. Trichomoniasis is a risk factor for HIV acquisition and shedding. Aetiology Although there are more than 100 species of this protozoan, only *T. vaginalis* parasitizes the human genital tract and has specific tropism for this environment. *Trichomonas tenax* can be found in the human oral mucosa and *Pentatrichomonas hominis* might be found in the human gastrointestinal tract. In women, *T. vaginalis* can be found in the vagina and the exterior cervix in over 95% of infections, but is recovered from the endocervix in 13%. The urethra and Skene's glands are also commonly infected. In men, the urethra is the most common site of infection but the organism has also been recovered from epididymal aspirates and semen. Epidemiology Epidemiological evidence suggests that *T. vaginalis* is transmitted almost exclusively by sexual intercourse, both during heterosexual intercourse and in sexual activity between female sexual partners. Although the organism can survive for many hours at room temperature if kept damp, there is only limited evidence that this pathogen is transmitted among household members in the absence of sexual exposure. No cyst form of the organism is known to exist. A very small proportion of female babies of infected mothers will become infected during birth, but this infection is transient and trichomoniasis discovered in a child should immediately raise the suspicion of sexual abuse. The recent availability of nucleic acid amplification tests (NAATS) for women and men has provided newer and more accurate data on the prevalence of infection. The US National Health and Nutrition Examination Survey conducted 2001–2004 projected that 3.1% of US women are infected. Rates were higher among minority populations. HIV infection and illicit drug use are risk factors for infection. *T. vaginalis* has been reported in 18–24% of women attending sexual health clinics in the United States of America and in 3–34% of women in four African cities. The epidemiology of this pathogen is less well understood among men, but has been reported in 3–20% of men attending sexual health clinics. In several developed countries, there has been a steady

decline in the incidence of trichomoniasis in the past few decades, but this has not occurred in less-developed countries nor in deprived inner-city areas in industrialized nations. Human trichomoniasis is becoming a disease of the underprivileged. Pathogenesis In vitro, *T. vaginalis* has a well-defined, contact-mediated, cytotoxic effect, but its relationship to pathogenesis in vivo is unknown. It activates complement and attracts neutrophils, which may kill the parasite but, in large numbers, might also contribute to the pathology. The organism produces several proteolytic enzymes which degrade genital tract mucins. Several potential *T. vaginalis* adhesions have been identified but, apart from its surface lipophosphoglycan, there is little evidence supporting a role in adhesion. Availability of the *T. vaginalis* genome sequence has allowed wider search for surface, soluble, and secreted proteins involved in host-parasite interactions. Clinical features In women, *T. vaginalis* can infect the vagina, urethra, and the Bartholin's and Skene's glands. From 10 to 50% of cases are asymptomatic but acute inflammatory diseases can occur, with copious and malodorous vaginal discharge, vulvovaginal soreness and irritation, dysuria, and dyspareunia. Trichomoniasis is significantly associated with purulent yellow vaginal discharge, vulvar itching, and colpitis macularis (strawberry cervix) detectable by colposcopy, with vulvar and vaginal erythema. The discharge fluctuates with time and, if untreated, might disappear spontaneously or persist for months or even years. Most men with trichomoniasis are asymptomatic, but the parasite is responsible for a significant number of cases of nongonococcal urethritis. Differential diagnosis In women, vaginal discharge syndromes including bacterial vaginosis, yeast vulvovaginitis, and trichomoniasis should be considered (see Chapter 9.4). Women who present with vaginal discharge, vulvar itching, and/or vaginal malodour might have no infection, or could have any combination of these common vaginal infections. In men, other causes of urethritis should be ruled out. An accurate diagnosis cannot be made based upon signs or symptoms elicited during the clinical evaluation. Trichomoniasis in women The most commonly used method for diagnosis is identification of the pathogen in vaginal (not endocervical) secretions examined under the microscope at $\times 400$ magnification. In clinical specimens or culture, *T. vaginalis* is a motile and round or oval flagellate, 10–13 μm long and 8–10 μm wide. Fixed and stained, it is about 25% smaller (Fig. 8.8.14.1). Diagnostic features include the jerky motility, undulating membrane, and microtubular rod (axostyle), which runs through the body and projects as a thin spine from the posterior end. Vaginal pH is usually elevated (>4.5) but can be normal. Microscopy is inexpensive and can be used as a bedside diagnostic test, allowing immediate treatment of infected people. However, its sensitivity is only 65–80% and it requires a microscope. Broth culture methods for detection of *T. vaginalis* have the advantage of greater sensitivity, but require up to 5 days' incubation (Diamond's TYM and the InPouch system). Rapid antigen tests can be performed within the clinic in a few minutes. Their sensitivity is 80% compared to culture, with the advantage of providing results during the clinic visit. The current gold standard for diagnosis is NAATS testing which can be performed on the same sample as collected for gonorrhoea

8.8.14 Trichomoniasis 1477 and chlamydia. These specimens include vaginal, endocervical, urine, or liquid-based Pap smears. Vaginal specimens can be self-collected. The sensitivity and specificity of NAATS testing exceeds 95%. Trichomoniasis in men Although currently male testing with NAATS has not been presented to the US Food and Drug Administration (FDA) for potential approval, NAATS is the diagnostic test of choice for men and has excellent sensitivity and specificity. The test can be run as an analyte specific reagent test in laboratories which have internally validated the assay in men. Urethral swab specimens or urine can be tested. Treatment 5-nitroimidazoles are the first line therapy. A single 2 g dose of oral metronidazole is most widely used. The alternative is 500 mg twice daily for 7 days. The latter is the recommended dose for women infected with HIV.

Metronidazole is not contraindicated in pregnancy. Recurrence occurs in 8 to 20% of women in the first month after therapy. About half the occurrences are attributed to reinfection by the same or a new sexual partner. Sexual partners must also be treated. Only the 7-day regimen has been extensively evaluated in men, in whom it appears as effective as in women. Treatment failures with metronidazole due to resistance occur 5–10% of the time. If reinfection can be ruled out, metronidazole for a longer treatment course can be tried. However, tinidazole should be considered due to its more favourable pharmacokinetic profile against *T. vaginalis*. There is no standardized dose for treating metronidazole resistant infections. One approach is 2 gm orally per day for 5–7 days. In cases of continued treatment failure, the combination of intravaginal paromomycin cream with high dose oral tinidazole has been reported to be successful. Because up to half of infected individuals are asymptomatic, the only way to reduce the population prevalence of this pathogen is through screening of individuals and providing treatment to individuals and their sexual partners. There is no effective vaccine against *T. vaginalis*.

Prognosis In most women who receive appropriate treatment, symptoms will resolve but they are at increased risk of becoming infected with *Trichomonas* in the future. About 50% of men will spontaneously clear their infections, but unless both sexual partners are treated, reinfection is common.

Complications Trichomoniasis in women was previously regarded as unpleasant but harmless; however, epidemiological studies have now linked it with a modest increase in the risk of heterosexual HIV transmission, and with complications in pregnancy.

Areas of uncertainty, controversy, and future developments Trichomoniasis has been linked with preterm birth, pelvic inflammatory disease (Chapter 9.8), and an increased risk of HIV. However, metronidazole treatment failed to reduce the risk of preterm delivery or acquisition of HIV in a limited number of previously conducted clinical trials which used culture for diagnosis as opposed to the currently available, more sensitive, NAATS. No study has yet documented that accurate diagnosis and treatment of trichomoniasis provides a long-term health benefit for men and women. In pregnant women, single-dose metronidazole treatment achieves parasitological cure, but one trial suggested an increased risk of preterm birth. Broader implementation of specific and sensitive screening tests and additional prospective studies of treatment should reveal whether routine screening and treatment of *T. vaginalis* reduces morbidity.

FURTHER READING Kissinger P, et al. (2013). Trichomoniasis and HIV interactions: a review. *Sex Trans Infect*, 89, 426–33. Meites E, et al. (2015). A review of evidence-based care of symptomatic trichomoniasis and asymptomatic *Trichomonas vaginalis* infections. *Clin Infect Dis*, 61 (Suppl 8), S837–48. Nye MB, et al. (2009). Comparison of APTIMA *Trichomonas vaginalis* transcription-mediated amplification to wet mount microscopy, culture, and polymerase chain reaction for diagnosis of trichomoniasis in men and women. *Am J Obstet Gynecol*, 200, 188.e181–7. Sena AC, et al. (2007). *Trichomonas vaginalis* infection in male sexual partners: implications for diagnosis, treatment, and prevention. *Clin Infect Dis*, 44, 13–22.

Fig. 8.8.14.1 Trichomonads in vaginal secretions (Giemsa stain). Copyright J. P. Ackers.

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